

PARASITES AFFECTING CNS

(AFRICAN TRYPANOSOMA)

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جامعة بنها الأهلية
مدينة العبور



PARASITES OF CNS

➤ **Taenia spp.:**

1. **Cysticercosis (*Taenia solium*).**
2. **Coenurosis (*Taenia multiceps*).**

➤ **Free living amoebae:**

1. ***Naegleria fowleri*.**
2. ***Acanthamoeba* spp.**

➤ **Trypanosomes:**

1. **African trypanosoma.**
2. **American trypanosoma.**

Objectives

By the end of this lecture the students should:

- ☐ List the species of trypanosomes.
- ☐ Illustrate morphology & life cycle of *Trypanosoma* spp.
- ☐ Mention clinical picture, diagnosis & treatment of sleeping sickness and chagas disease.

❑ Trypanosoma is **flagellated protozoa**.

❑ Protozoa are unicellular micro-organisms able to perform all vital functions.

TRYPANOSOMA

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graph TD; A[TRYPANOSOMA] --> B["African Trypanosoma  
(Trypanosoma brucei)"]; A --> C["American Trypanosoma  
(Trypanosoma cruzi)"]; B --> D["Trypanosoma brucei  
gambiense"]; B --> E["Trypanosoma brucei  
rhodesiense"];
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African Trypanosoma
(Trypanosoma brucei)

American Trypanosoma
(Trypanosoma cruzi)

Trypanosoma
brucei
gambiense

Trypanosoma
brucei
rhodesiense

I-AFRICAN TRYPANOSOMES

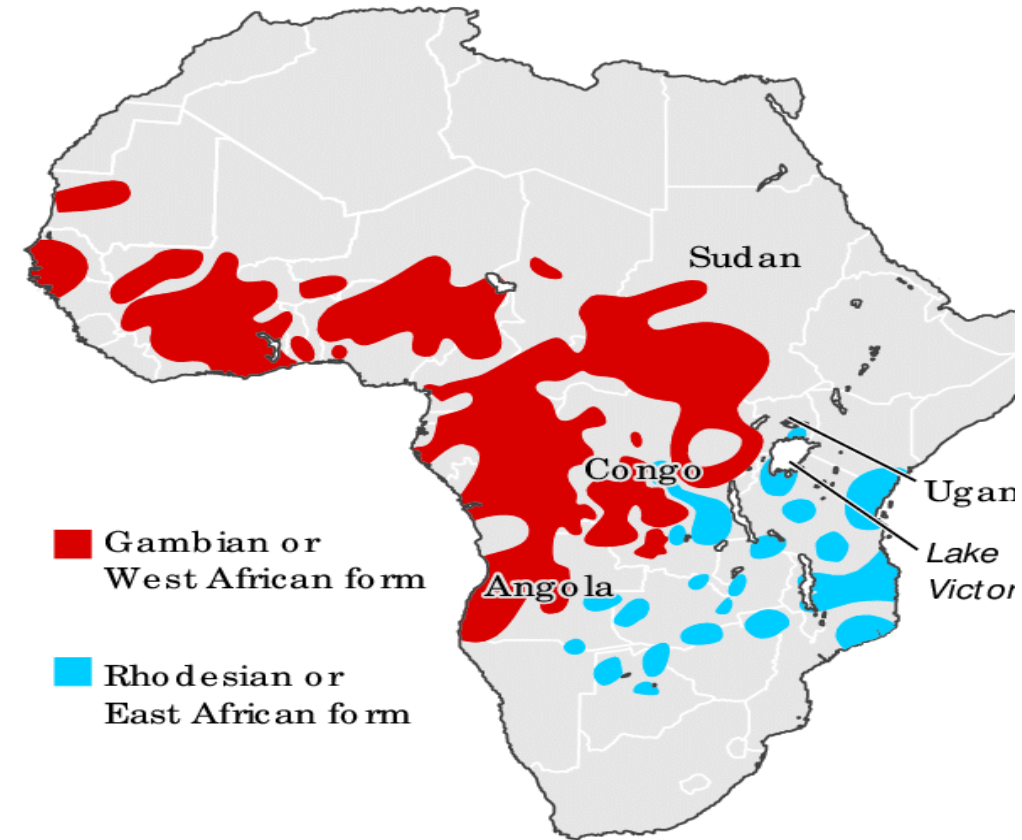


1. *Trypanosoma gambiense*

Disease:

Gambian trypanosomiasis, chronic african trypanosomiasis, west and central African sleeping sickness.

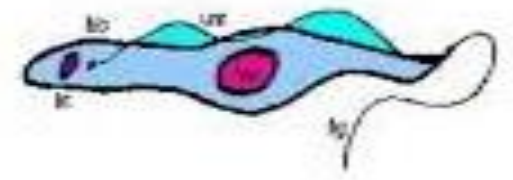
Geographical distribution: West and central Africa.



Morphology

1) Trypomastigote (polymorphic) form:

- It occurs in **blood** of mammalian hosts (including man).
- The body is spindle and kinetoplast is posterior to the nucleus with undulating membrane with a free flagellum.
- These are of different sizes:
 - (i) The short stumpy forms, 15 to 25 μm long.
 - (ii) The elongate slender forms, 20 to 40 μm long.



2) Epimastigoste (Crithidia) form:

- It occurs in the **vector & culture**.
- The nucleus is posterior to the kinetoplast and there is an undulating membrane with a free flagellum. .



Life cycle



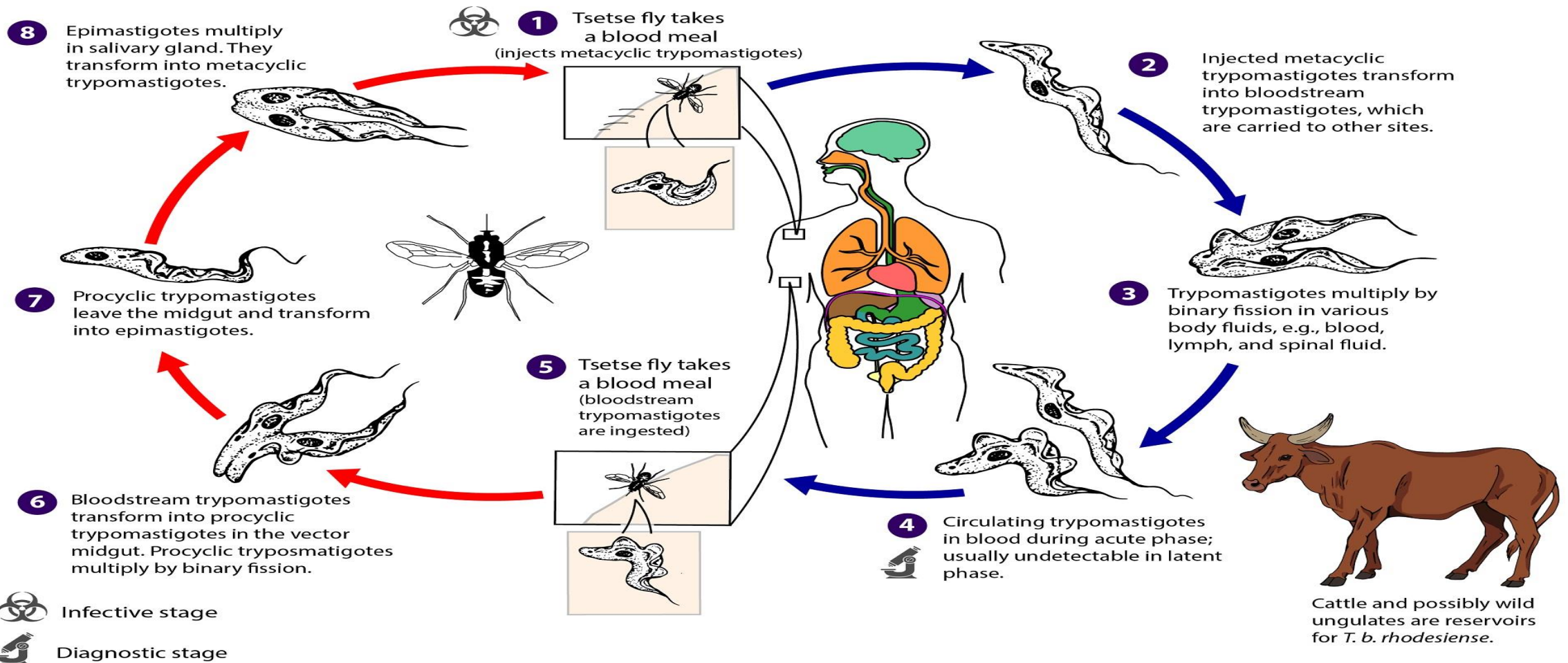
African Trypanosomiasis

Trypanosoma brucei gambiense & *Trypanosoma brucei rhodesiense*



Tsetse Fly Stages

Mammalian Stages



1- Habitat: All tissues of the vertebrate host.

2- Vertebrate host: :Man, Pigs, dogs and goats.

3- Vector: *Glossina palpalis*(tsetse fly).

4- Infective stage: Metacyclic form (Anterior station).

5-Mode of infection:

- 1) The infective stage passes with the vector saliva during blood meal.
- 2) Congenital infection is rare, because in trypanosomiasis most pregnancies end in abortion.

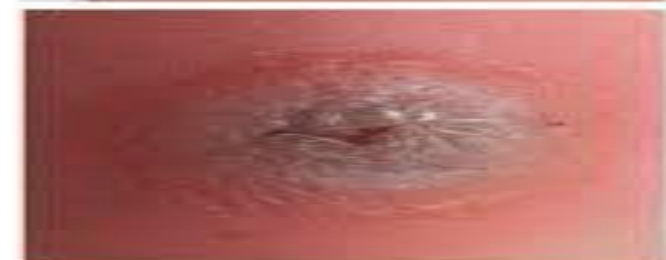
Pathology

- ❑ **At the site of the bite:** The organisms multiply in the subcutaneous tissue producing a local inflammatory lesion, the trypanosome "**chancre**".
- ❑ **Invasion of blood and lymphatics** cause generalized **toxaemia and enlarged lymph glands**. The key pathologic lesion is **small vessel vasculitis** and increases permeability leading to edema in any organ.
- ❑ **Sleeping sickness stage:** Brain invasion with **peri-vascular infiltration** results in generalized **meningoencephalitis**. The **morula cells** (altered plasma cells) found in CSF is typical for sleeping sickness, but it may be found in other conditions.

Clinical manifestations

1. Chancre:

- A trypanosomal chancre may appear around 48 hours after the tsetse bite.
- Itchy, painful, inflammatory reaction at the site of the bite.
- The chancre is an indurated (hardened) red nodule, 2 to 5 cm in diameter.
- A central necrotic dark-coloured crust may form before the chancre desquamates within 2 to 3 weeks, leaving no trace.



• 2. Hemolymphatic stage:

This stage may be asymptomatic and infection may be self-cure within few months.

- Remittent, irregular fevers with night sweats.
- Headache, malaise, anorexia and weight loss.
- There may be delayed pain sensation, "**Kerandel's sign**".
- The liver and spleen may be enlarged.
- Edema is frequent in the form of peripheral edema, pulmonary edema, or pericardial effusion.

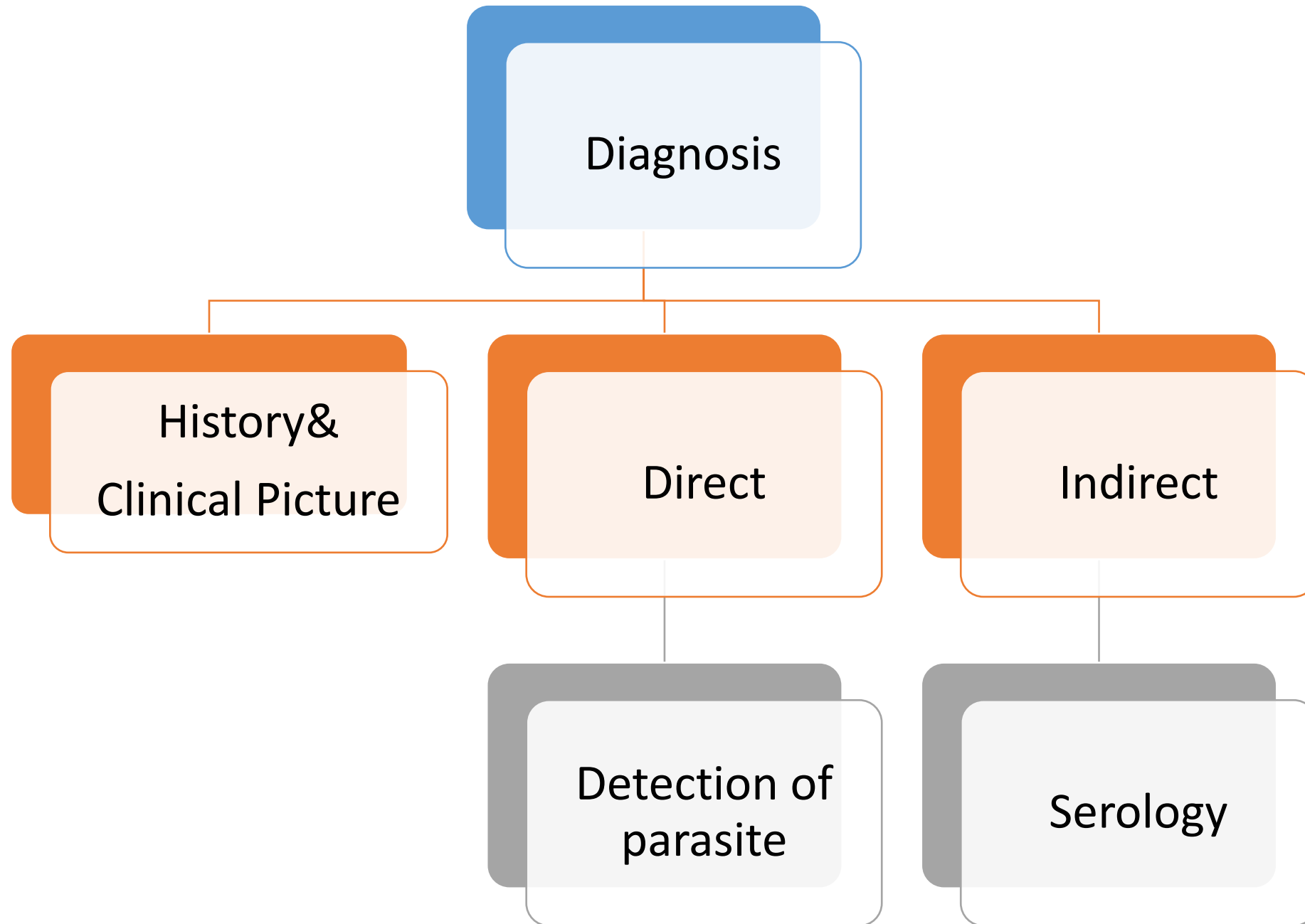
- Lymph nodes enlargement occurs in about 75% of patient.
- Enlargement of nodes in the lower posterior cervical triangle is called **"Winterbottom's sign"**.



3. Meningoencephalitic stage:

- There is steady progressive apathy, confusion, fatigue and coordination loss.
- In terminal phase, the patient becomes emaciated, progressing to coma and death.



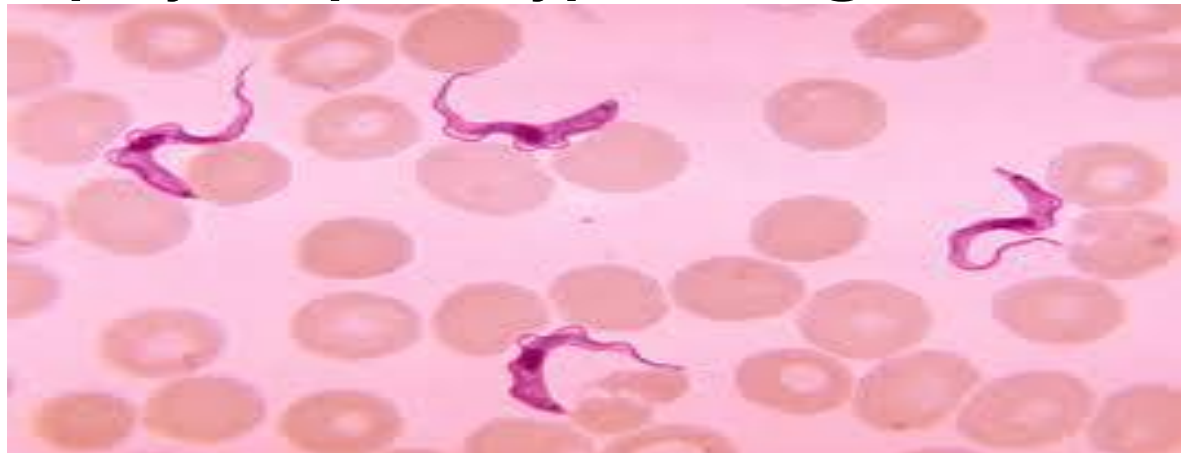


Diagnosis

1. Clinical picture

2. Direct (Demonstration of the parasite):

- Specimen: blood, CSF, lymph node aspirates, and sternum bone marrow.
- Microscopic examination polymorphic trypomastigote.



N.B: C.S.F. examination:

Increased total cells count (more than 5).

Elevated protein (above 40 mg/dl).

High **IgM** titer.

Presence of **morula cells**.

3. Indirect (Serological tests):

The serologic tests become positive from about the **12th day** of infection.

Treatment

Depend on the stage of the disease

1. Hemolymphatic stage:

Suramin (Antrypol) and **pentamidine** isothionate, cannot penetrate the blood-brain barrier.

2. Meningoencephalitic stage:

Melarsoprol (Arsobal) is capable of penetrating the blood-brain barrier.

Control and prevention

1. Directed to treatment of patients.
2. Destruction of *Glossina* and its breeding places.
3. Chemoprophylaxis (Antrypol).

2. Trypanosoma rhodesiense

- ❑ *T. rhodesiense* is morphologically and biologically similar to *T. gambiense* except for certain differences:

1. Geographical distribution:

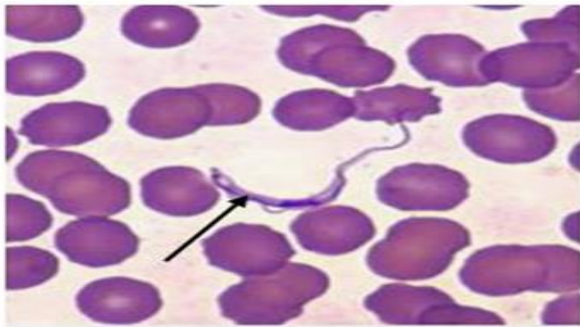
Eastern part of Africa.

2. Life cycle:

Trypanosoma brucei causing Sleeping Sickness

West Africa

T. brucei gambiense



Less plentiful

Cannot live in lab animals

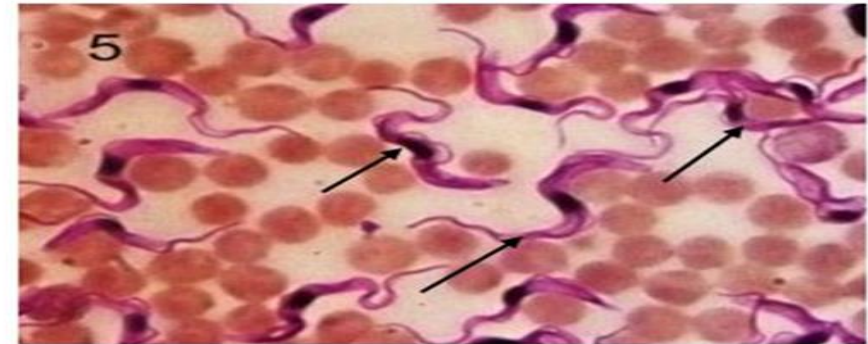
Reservoir host:

goats, cattle & pigs

Transmitted by: *G. palpalis*

East Africa

T. brucei rhodesiense



More plentiful

Can live in lab animals

Nucleus is shifted posteriorly



Reservoir host:

wild game animals

Transmitted by: *G. morsitans*

3. Clinically:

- *T. rhodesiense* has a shorter incubation period and **rapid C.N.S. involvement** with an **acute** form of the disease which ends fatally in untreated cases.
- **Lymph nodes** involvement is **less pronounced**, and Winterbottom's sign may be absent.
- The patient is more anemic and more likely to develop myocarditis.

4. Diagnosis:

Serodiagnosis may be difficult with many **false negative** results in early diagnosed cases, because infection with *T. rhodesiense* is so fulminant, the immune response with **humoral antibody will be less** than that seen in *T. gambiense*.

Infective stage of african trypanosomiasis is ...

- a. Trypomastigote
- b. Amastigote
- c. Metacyclic form
- d. Promastigote

In african trypanosomiasis local inflammation lesion is called

- a. chancre
- b. edema
- c. winterbottom sign
- d. kerandell sign

In african sleeping sickness with cns involvement we use in treatment :

- a. suramin
- b. Melarsoprol
- c. pentamidine
- d. Amphotericin B



Diagnostic stage of african trypanosomiasis is ...

- a. Trypomastigote
- b. Amastigote
- c. Metacyclic form
- d. Promastigote

Vector of african trypanosomiasis is ...

- a. *Anopheles* mosquito
- b. *Culex* mosquito
- c. Sand fly
- d. Tsetse fly



- Discuss diagnosis and prevention of African sleeping sickness.
- Evaluate treatment of **african trypanosomiasis**

Reference

Henry's Clinical Diagnosis and Management by Laboratory Methods MEDICAL PARASITOLOGY

<https://0810fk5ao-1104-y-https-www-clinicalkey-com.mplbci.ekb.eg/#!/browse/book/3-s2.0-C20180017521>

Richard A. Mcpherson, Matthew R. Picnic (2021)

Henry's Clinical Diagnosis and Management by Laboratory Methods, Twenty Fourth Edition chapter 65
MEDICAL PARASITOLOGY Elsevier June 1, 2021

Recommended :

2-Textbook_of_MEDICAL_PARASITOLOGY

[https://www.academia.edu/44759301/Textbook of MEDICAL PARASITOLOGY Textbook of MEDICAL PARASITOLOGY](https://www.academia.edu/44759301/Textbook_of_MEDICAL_PARASITOLOGY_Textbook_of_MEDICAL_PARASITOLOGY)

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THANK YOU

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